



## **Sensory Neuron Sensitivity In *Drosophila Melanogaster* Is Regulated By Translation Initiation Factors**

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### **Abstract**

Millions of Americans suffer from chronic pain each year, and pain symptoms are the leading cause of hospitalization and long-term disability. A significant hurdle for our understanding of pain syndromes is an incomplete appreciation of the cellular, molecular, and genetic factors that contribute to their pathogenesis. To understand the pathogenesis of chronic pain, we must identify the mechanisms that control the sensitivity of somatosensory neurons under basal conditions as well as the plasticity mechanisms that allow somatosensory neurons to become sensitized following injury or environmental insult. The induction and maintenance of long-term plastic changes in neurons requires precise regulation of gene expression. One mechanism for accomplishing this is tight regulation of protein synthesis. My lab investigates these processes using the fruit fly, *Drosophila melanogaster*, as a model organism. Our work focuses on the overarching hypothesis that precise regulation of translation initiation controls sensory neuron sensitivity. We have recently found that translation initiation factor proteins are required to regulate the sensitivity of larval sensory neurons of *Drosophila* to noxious thermal stimuli. Additionally, these factors are also required for changes in sensory neuron sensitivity after the larvae experience tissues damage. These results suggest a model where translation initiation factors regulate the synthesis of proteins required for baseline sensory neuron function and also for changes in sensory neuron function following injury. Our ongoing work investigates the cellular and molecular mechanisms by which these translation initiation factors are activated during development and following injury to maintain appropriate sensory neuron sensitivity.

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